Update of 2002 Michigan Surveillance and Response Plan for Chronic Wasting Disease (CWD) of Free-ranging and Privately-owned/Captive Cervids[*]

5 March 2012

Background and rationale: Michigan's original Surveillance and Response Plan for Chronic Wasting Disease (CWD) of Free-ranging and Privately-owned (PO)/Captive[†] Cervids (POC) [hereafter, the Plan] was finalized in August 2002. Its development was set in motion by the initial discovery of CWD in Wisconsin in February 2002. At that time, what was known about CWD was limited to developing research on the outbreaks in mule deer (*Odocoileus hemionus*) and Rocky Mountain elk (*Cervus elaphus nelsoni*) in northern Colorado and southeastern Wyoming. There was no scientific information on how the disease might behave in free-ranging white-tailed deer (*Odocoileus virginianus*) populations in eastern North America, which are typically present at much higher densities. In the decade that has since passed, a large amount of research has been published which much more fully describes CWD: what causes it, how it is transmitted, how environmental conditions affect its spread, its infectiousness to other species, and public opinions concerning the disease and how it should be managed. Moreover, invaluable case studies now exist which document agency attempts to manage the disease, and responses to those attempts by policymakers and the public. Michigan's CWD Response Plan specifically was also subjected to expert external scrutiny.[‡] Incorporation of this knowledge and experience is consistent with the principles of adaptive natural resource management.

On reviewing Michigan's 2002 Plan in 2012, it remains largely both scientifically valid and defensible. However, recent experience with CWD in other states (and with other diseases such as bovine tuberculosis (bTB) in Michigan) has shown that it can be practically difficult to implement response plans that are insufficiently targeted towards the specific locations in which diseased deer are present.

This update of Michigan's Plan implicitly recognizes these practical difficulties and strikes a balance in which the strongest response that can be practically implemented is coupled with flexibility to adapt that response to the specifics of situations in which CWD is detected in either captive or free-ranging cervids.

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[*] This document borrows from Minnesota's Chronic Wasting Disease Response Plan; the Michigan DNR Wildlife Division is indebted to Dr. Michelle Carstensen, Minnesota DNR, for sharing it.

[†] Under Michigan law, farmed deer, elk and other cervids are referred to as "privately-owned," rather than by the commonly-used term "captive", to distinguish them from free-ranging, publicly-owned animals. Both "privately-owned" and "captive" are included here in the interest of clarity for a diverse audience.

[‡] *Final Report, Michigan Chronic Wasting Disease Task Force*, dated October 15, 2003, available at http://www.michigan.gov/documents/emergingdiseases/CWDTaskForceFinalReport_382672_7.pdf ; and Ducrocq, J. et al. *Final Report: A Systematic Review of Michigan's Policy For CWD Prevention, Detection and Control*, dated 8/2007, available at http://www.michigan.gov/documents/emergingdiseases/CWD_Review_Michigan_382615_7.pdf .

Scientific advances related to CWD since the 2002 Plan: A large, diverse body of scientific research has accumulated since the 2002 Plan. A concise review is presented in Appendix A.

Guiding principles drawn from the current state of the science

1. CWD is an infectious prion disease, and claims to the contrary are not scientifically credible.
2. CWD is transmitted between animals by direct contact with infectious saliva, respiratory aerosols, urine, and feces. Infected animals are infectious for other animals before they appear sick. Infected animals inevitably succumb, although the amount of time that takes to happen can vary from months to years.
3. CWD is also transmitted indirectly from contaminated items in the environment such as soils where it persists for decades. Where the disease becomes established, environmental contamination likely drives CWD outbreaks perpetually, and may be the most critical factor limiting their control. Substantial environmental contamination with CWD may effectively define the threshold for when the disease is 'established'.
4. There is essentially no evidence that CWD can infect humans. While recognizing that some members of the public may perceive it as a risk, management of CWD need not assume it is a substantial threat to human health.
5. As CWD prevalence and perceived threats to human health increase, abandonment of hunting in infected areas may seriously limit the most practical approaches by which agencies may control the disease, and have a potentially catastrophic impact on hunter recruitment.
6. The public supports lethal management to control wildlife disease when that control achieves desired ends. Non-hunters are largely unconcerned with CWD and its management. Hunters are mainly concerned with the effect of CWD on deer hunting and the safety of venison for human consumption.
7. CWD surveillance based solely on testing of hunter-harvested cervids has a low probability of detecting the disease, and may be biased. By the time cervids with clinical disease are detected, the prevalence of CWD in the population is likely to be over 1%, and the disease already effectively established.
8. Effective CWD management relies on preventing establishment of the disease in the first place. Once CWD is established in an area, all methods tried to date have failed to eradicate the disease. Current evidence suggests that in those situations, cervid density reduction is no longer likely to be helpful. Nonetheless, density reductions in surrounding areas may help limit geographic spread.
9. Density reductions should target entire family groups (does and their fawns) to minimize the probability of disease persistence, and yearling bucks to minimize the probability of disease spread via dispersal. Hunter harvest decisions depend most heavily on personal attitudes and are relatively unaffected by agency educational efforts. For these reasons, agency culling is likely to be more effective for controlling CWD than hunter harvest.
10. Management practices that increase biological carrying capacity (such as supplemental feeding by humans) may cause CWD to persist and spread, just as

they do with other diseases such as bovine tuberculosis. Alternative strategies for allowing supplemental feeding to continue in a restricted manner do not mitigate the potential for CWD transmission.

11. Once established, CWD outbreaks (and the substantial costs of their management) can be expected to last for decades.

Surveillance Plan for Free-ranging Cervids (supersedes section II.A of the 2002 Plan). The fundamental goals of Michigan DNR's CWD surveillance remain unchanged from 2002: testing of free-ranging white-tailed deer, elk and moose to determine the presence/absence and extent of the disease. To the date of this writing, more than 34,000 deer, 1600 elk and 70 moose have been tested statewide; all have been CWD negative.

Because of 1) the costs associated with active surveillance (i.e., testing hunter-harvested deer), 2) its documented potential for bias and 3) the failure to detect CWD in free-ranging cervids to date despite extensive, statewide active surveillance, it is anticipated that increasing emphasis will be placed on targeted surveillance on an ongoing basis, with active surveillance employed following diagnosis of any CWD positive free-ranging or captive cervid. Where CWD testing will occur and how many animals will be sampled will depend upon the situation in a particular year.

Screening of free-ranging cervids for CWD will be via enzyme linked immunosorbent assay (ELISA) testing of medial retropharyngeal lymph nodes at the DNR's Wildlife Disease Lab in Lansing. Confirmation of ELISA suspects will be via immunohistochemistry (IHC) testing at Michigan State University's Diagnostic Center for Population and Animal Health (DCPAH); IHC-positive tests will shipped to the U.S. Department of Agriculture's National Veterinary Services Laboratory (USDA-NVSL) for final confirmation as CWD-positive.

Surveillance Plan for Captive/Privately-Owned Cervids (POCs) (supersedes section II.B of the 2002 Plan). The Michigan Department of Agriculture and Rural Development (MDARD) will conduct surveillance on POC herds. Michigan has approximately 450 privately owned cervid facilities numbering about 25,000 animals. Through routine surveillance, in August 2008, one captive white tailed deer was diagnosed with CWD in Michigan. The herd was depopulated, with no other positives detected in the herd, or on any traces to and from the herd. .

On April 27, 2002 a moratorium was placed that bans the importation of cervids into Michigan. In 2011 guidelines were developed to allow exemptions to the moratorium on a case by case basis. A copy of the importation exemption requirements is listed in Appendix B.

1. CWD Mandatory Surveillance
    a. Compliance with operational standards, including:

i. Perimeter fence requirements;
ii. Animals identified by two approved methods;
iii. Mandatory death reporting;

b. Surveillance testing of animals over 12 months of age that die, are sick, and a percentage of culls and slaughter animals;

c. Positive diagnosis is based on testing proper segments of the brain, and retropharyngeal lymph nodes

i. Initial suspect positive is determined at a National Animal Heath Laboratory (NAHLN)
ii. Confirmatory diagnosis is made at National Veterinary Services Laboratory (NVSL)

2. Voluntary CWD Certification Program

a. Compliance with operational standards, including:

i. Perimeter fencing requirements;
ii. Record keeping requirements;
iii. Animal movement restrictions;
iv. Animals identified by two approved methods;
v. Mandatory death reporting;

b. Surveillance testing of all animals over 12 months of age that die;

c. Annual verification of animal inventory by state veterinarian;

d. Positive diagnosis is based on testing proper segments of the brain, and retropharyngeal lymph nodes

i. Initial suspect positive is determined at a National Animal Heath Laboratory (NAHLN)
ii. Confirmatory diagnosis is made at National Veterinary Services Laboratory (NVSL)

e. Herd status based on years of surveillance;

f. This is a six-year plan to achieve CWD certified status for a herd.

3. All POC facilities are regulated under Public Act 190 of 2000. This act is regulated by the Michigan DNR. Animal health related programs are regulated by MDARD.

a. Mandatory registration of all facilities;

b. Requirements for minimum fence heights and acceptable fence materials;

c. Mandatory fence inspection;

d. Mandatory yearly submission of fence inspection reports;

e. Mandatory record keeping;

1. Maintaining records of all additions to herd;
2. Maintaining records of all losses from the herd;
3. Maintaining records of all health certificates and test results;
4. All cervids must be officially and individually identified;

f. Mandatory yearly submission of animal inventories;

g. Recovery protocol for escaped cervidae;

h. MDARD/DNR maintains a database of all cervid facilities with location, size, type, contact number, and number of animals present;

i. Instate movement restrictions based on registration class.

4. CWD is a reportable disease. Per 1998 PA 466, any owner, veterinarian, or member of the public who *suspects* CWD must report it to the MDARD immediately. MDARD veterinarians who are trained in the diagnosis of the disease will be dispatched to do the follow-up on the report.

Response Plan for Free-ranging Cervids (supersedes section III.A of the 2002 Plan). The fundamental goals of Michigan DNR's CWD response remain largely unchanged from 2002: 1) identification and response to limit further transmission of the disease; and 2) eradicate CWD from both PO/captive and free-ranging cervids if the results of surveillance suggest that is likely to be achievable.

If CWD is identified in either a PO/captive or wild cervid, either within the boundaries of Michigan or within a biologically-relevant distance of the Michigan border, the following measures should be implemented as rapidly as possible:

1. Complete a population survey in the area where the CWD-positive cervid was detected to estimate free-ranging cervid species presence, density and distribution.
2. Establish a CWD Management Zone, the size of which will depend on the location, species, captive/wild status and distribution of infected cervids as well as the density, distribution and seasonal movements of the local wild cervid population(s).
3. Implement a deer feeding and baiting ban, which at a minimum should include the entire CWD Management Zone.
4. Prohibit the movement of both captive and free-ranging cervid carcasses and parts from the CWD Management Zone.
5. Intensify surveillance efforts in free-ranging cervids in the CWD Management Zone, with mandatory check and CWD testing of all cervids taken in the Zone.

Following these initial steps, the prevalence and spatial distribution of CWD will be assessed to advise subsequent response actions.

1. *Identification of an infected PO/captive cervid facility*: The primary objective of DNR response efforts will be to determine if free-ranging cervids in the vicinity of the PO/captive herd are also infected with CWD and, if so, the magnitude and geographic extent of that infection.

   In the event an infected PO/captive cervid is identified, the following measures should be implemented as rapidly as possible:

   a) Geographic Information Systems (GIS) methods will be used to map the location of the infected PO/captive cervid (index case). A *five*-mile radius circle will be defined around the index case. At a minimum, any county the boundary of which is intersected by that radius will be defined as part of the CWD Management Zone. If the results of the local population survey or credible scientific evidence suggests that cervids from within the radius are likely to move beyond these

Management Zone boundaries, those boundaries should be expanded accordingly.

b) Surveillance goals (i.e., sample size, geographic distribution, age/sex distribution, etc.) should be established for the CWD Management Zone based on results of the population survey, the current state of the science as related to CWD control, and consultation with disease control staff in other states/provinces.

c) Surveillance should commence. If sufficient financial and personnel resources allow, expanded efforts to obtain cervids for testing via both targeted surveillance and testing of opportunistically obtained animals (e.g., roadkills) should occur.

   Presumably, in addition to these sources of test animals, free-ranging cervids for testing will need to be obtained via active surveillance. Sampling methods should depend on the date of diagnosis of the index case in relation to the next cervid hunting season.

   1. If the next season is to commence within less than six months, hunting opportunities should be liberalized by expanding seasons and increasing available licenses or permits. Those harvested animals should be used as the primary means of determining CWD prevalence and distribution. Regulations should mandate testing of any cervid harvested within the Management Zone by presenting either the animal's head or the entire carcass at a DNR check station.
   2. If hunters do not kill a sufficient number of cervids to provide an adequate sample, DNR should collect additional samples via designating special hunts, landowner shooting permits, agency-directed culling and/or other methods as deemed necessary.
   3. If there are more than six months between diagnosis of the CWD-positive and the next hunting season, DNR should collect needed samples via designating special hunts, landowner shooting permits, agency-directed culling and/or other methods as deemed necessary. These efforts should be followed by liberalized hunting opportunities, and active surveillance of hunter-harvested cervids should occur during the autumn hunting seasons.
   4. Locations of CWD-positive animals identified during surveillance should be evaluated and the boundaries of the CWD Management Zone adjusted as necessary.
   5. Active surveillance via hunter harvest and/or other methods should become an annual occurrence designed to monitor changes in CWD prevalence and spatial distribution for a period deemed epidemiologically appropriate.

2. *Identification of an infected free-ranging cervid*: Whether identified as a result of testing around a CWD-infected PO/captive cervid facility as above, or as a result of ongoing targeted surveillance of suspect free-ranging cervids, the primary objective of DNR response efforts should be to rapidly determine the prevalence and

geographic extent of CWD infection in the wild population, followed by rapid decision making by policymakers of the course of action to be taken.

In the event an infected free-ranging cervid is identified, the following measures should be implemented as rapidly as possible:

a) Geographic Information Systems (GIS) methods will be used to map the location of the infected cervid (index case). A *ten*-mile radius circle will be defined around the index case. At a minimum, any county the boundary of which is intersected by that radius will be defined as part of the CWD Management Zone. If the results of the local population survey or credible scientific evidence suggests that cervids from within the radius are likely to move beyond these Management Zone boundaries, those boundaries should be expanded accordingly, recognizing that circumstances may warrant a stronger response.

b) Establishment and execution of intensified surveillance, as outlined for *Identification of an infected PO/captive cervid facility*, points 1.b & c, above.

Once surveillance data are compiled and presented to policymakers, decisions must be made concerning the necessity, nature and extent of response actions. These decisions should be made expeditiously. That decision making process[1] should be informed by:

- the results of surveillance,
- the current state of the biological science
- recognition that CWD surveillance alone does not constitute a meaningful or useful response
- the likely costs and consequences of both action and inaction.

Once infectious diseases have become established in free-ranging wildlife populations, they may be extremely difficult or practically impossible to eliminate. Several states and provinces have discovered CWD and implemented management plans varying from complete inaction to aggressive attempts at eradication. None has yet been successful in eliminating the disease from wild cervids once it has become established. Owing to the importance of environmental contamination in maintaining and transmitting CWD, the key factor is the initial prevalence and distribution of infection at the time it is detected in the free-ranging cervid population.

For example, when Wisconsin first discovered CWD in wild deer in 2002, it was assumed to be a recent introduction, and management initially focused appropriately on disease eradication. However, subsequent surveillance revealed that the disease was already widespread by the time of discovery[2], making the prospects for eradication very unlikely. After spending approximately $30 million to combat the disease, CWD is endemic in southern Wisconsin, increasing in prevalence at a rate of 4% per year, with the affected geographic area continuing to expand. While the effect of CWD on the free-ranging deer population in Wisconsin is not expected to be markedly detrimental over

the next decade, research from Colorado and Wyoming has already demonstrated the CWD is likely to reduce deer populations. In the Boulder, Colorado area, annual survival of CWD-infected adult mule deer was markedly lower (53%) than uninfected adults (82%). In the ~23 years since CWD was recognized in the area, 41% of bucks (50-80% of prime age 3 & 4 year old bucks) & 20% of does were CWD-infected, and the population experienced a 45% decline in abundance[3].

In contrast, New York discovered CWD in 2005 in the free-ranging deer population surrounding a CWD-positive POC facility[4]. Initial surveillance found only one positive free-ranging deer, a prevalence of <0.1%, and subsequent surveillance has thus far failed to detect additional infected deer in the wild. The swift, aggressive response (which included agency culling and enhanced opportunistic, targeted, and hunter-harvested surveillance efforts) taken by the wildlife agency appears to have occurred prior to CWD becoming established in the population. While it is still too early to determine if CWD has been eradicated entirely, New York's response may have at least limited its spread.

Thus, both the current state of scientific research and the experience of other states support an aggressive approach to prevent establishment of CWD in the wild population as the only course of management likely to be successful. Once it is allowed to become established, eradication of CWD is not a realistically achievable, or cost effective, management objective. Containment then becomes the default option, one for which success is far from certain.

With this document, like Minnesota, Michigan DNR recommends the response to CWD diagnosis in free-ranging cervids be updated to an adaptive management strategy, allowing flexibility to alter disease management activities depending on the state of the disease at the time of discovery, the effectiveness of the methods applied, future research results, and the willingness of policymakers and the public to implement the control measures supported by scientific evidence. This update to Michigan's 2002 Response Plan provides for evaluation and monitoring of the prevalence and geographic extent of CWD infection, with management measures subsequently implemented based on evaluation of testing results, and in the long term, the effectiveness of the strategies employed.

<u>Response Plan for POCs (supersedes section III.B of the 2002 Plan)</u>. The MDARD CWD response efforts will entail:

A.

1. If CWD is diagnosed in free-ranging or POC, MDARD will activate an Incident Management Team (IMT) with MDNR under unified command procedures to handle the animal disease event.
2. The IMT will follow procedures as outlined in the National Incident Management System.
    a. Each department will identify Agency Administrators and assign one (1) Incident Commander from each department to the event.

i. Lead incident commander for the event will be chosen by agency administrators based on:
    1. Which department has lead authority (e.g. wildlife response vs. POC response);
    2. Experience;
    3. Incident Command System training.
ii. The remainder of the IMT will be chosen by the incident commanders from members of both agencies who have appropriate ICS training.

b. Objectives of the IMT are to create an Incident Action Plan (IAP) that may include but is not limited to:
    i. Safety of all responders, public, and appropriate animal handling response;
    ii. Coordinate disease response between MDNR, MDARD, USDA, and other external stakeholders to contain and eliminate disease while allowing continuity of business if appropriate

c. Recommended tactics for the Operations Unit may include, but are not limited to:
    i. CWD diagnosed in free-ranging cervid (one positive animal in 15-mile radius).
        1. Define a 15-mile radius around each positive case and identify all POC facilities.
        2. Biannual herd records inspection by personnel, and an annual fence inspection by MDNR personnel. Indemnity may be paid for these animals if funding is available.
            a. CWD testing of all death losses of animals twelve (12) months and older.
            b. Surveillance will continue for 60 months.
    ii. CWD diagnosed in free-ranging cervids (two or more positive animals within a 15-mile radius).
        1. Define a 5-mile radius surveillance zone around each positive case and identify all POC facilities.
            a. POC – Perform an epidemiological investigation to determine possible exposure of POC to infection.
            b. POC – If feasible, depopulate with indemnity, if available, all POC over twelve (12) months of age and older, and test for CWD.
            c. POC – If depopulation is not possible due to economics or the number of positive cases present:
                i. Quarantine facility;
                ii. Perform epidemiological investigation to determine possible exposure of POC to CWD.
                iii. Biannual herd records inspection by MDARD personnel, and an annual fence inspection by MDNR personnel with removal and testing of

any suspect animals for CWD. Indemnity will be paid for these animals if available.

iv. CWD testing of all animal death losses twelve (12) months and older.

v. Surveillance will continue for sixty (60) months.

1. Define a 15-mile radius around each positive case and identify all POC facilities between the 5-mile radius and the 15-mile radius.

a. Perform an epidemiological investigation to determine possible exposure of POC to CWD.

b. Biannual herd records inspection by MDARD personnel, and an annual fence inspection by MDNR personnel with removal and testing of any suspect animals for CWD. Indemnity may be paid for these animals if available.

c. CWD testing of all death losses of animals twelve (12) months and older.

d. Surveillance will continue for sixty (60) months.

iii. CWD diagnosed in POC herd

1. The state veterinarian's office (in conjunction with a CWD epidemiologist) shall conduct a complete epidemiological investigation to determine the specific cause, source of disease, population exposed, and population infected.

2. Quarantine the facility.

a. Depopulate the herd. Follow MDNR recommendations for appropriate depopulation process. Provide indemnity if available.

b. CWD test all animals twelve (12) months of age and older.

c. Appropriate disposal of suspected prion infected carcasses

i. Disposal methods may include incineration, alkaline digestion (or similar process), landfill only with special designated vault.

ii. The positive herd premises shall be cleaned and disinfected according to directions prescribed by the state veterinarian that are designed to minimize the spread of CWD. The

facility will be released from quarantine with an agreement between MDARD, MDNR, and the producer that the facility will be utilized for non-cervid livestock only.

3. Trace forward of exposed animals
    a. The facility will be placed under quarantine.
        i. Removed exposed animal, with indemnity if available, and test for CWD.
        ii. If the exposed animal(s) is positive, the entire herd is positive.
        iii. If the exposed animal(s) is negative, routine CWD surveillance (test of death losses over twelve (12) months of age) will continue for sixty (60) months.
4. Trace back of exposed animals
    a. Quarantine the herd for sixty (60) months from the last case traced back to the herd.
    b. Monthly inspection of the herd by state or federal personnel with euthanasia and testing of any suspect animals. Indemnity will be paid for these animals if available. Disposal of animals must follow a protocol set by the state veterinarian.
    c. Surveillance (testing all death losses over twelve (12) months of age) will continue for sixty (60) months.

iv. Recommended biosecurity measures will be addressed using the latest information available. See Appendix B for more details.

<u>Education/Outreach/Communications on Response Activities (as in section III.C of the 2002 Plan)</u>.– In the event of a CWD confirmation in Michigan, communication will play a critical role. The state's handling of the situation in the first 24 hours and the ensuing 10 days will have a lasting impact on public perception of the state's ability to address and control the disease. The MDNR and MDARD will designate limited knowledgeable spokespeople and work through agency Public Information Officers (PIOs) to provide the most up-to-date information to the media, public, and other non-governmental entities.

Regardless of whether it is in a free-ranging or PO/captive cervid population, confirmation of a CWD infection in Michigan will involve MDARD and MDNR in a series of actions and communications. Developments in other states with CWD have shown that ambitious depopulation plans can be controversial. Agency officials from MDNR and MDA must outline a coordinated effort to address the situation, and maintain continual public communications to explain and update actions and goals. Key communication activities which will need to be undertaken include, but are not limited to:

1. Security: Notification will take place upon official laboratory confirmation of CWD-positive test results.

2. Notification: Interagency communication will begin immediately, with notice proceeding up the divisional chain of command to each Department Director. The Directors will inform the Governor's press, legislative, and policy offices; the Natural Resources Commission (NRC); the Commission of Agriculture; and the Director, Department of Community Health.

3. A meeting of key representatives from MDNR, MDARD, the Governor's office, the NRC, and the Commission of Agriculture will be arranged as soon as possible to arrange a public announcement of the discovery and implement disease response strategies.

4. A media advisory will be issued following the meeting to announce a press conference. The press conference will be held in Lansing at one of the state buildings (Capitol, Romney, Mason, Constitution Hall, etc.).

5. Agency directors or designees will make calls to key constituency/stakeholder groups, including counterparts in other Great Lakes states, appropriate federal agencies, legislators, local municipality officials where the discovery is made, and university collaborators, to inform them of the CWD confirmation and impending announcement.

6. The MDNR and MDARD Directors, and possibly the Governor, will confirm the presence of CWD in Michigan and outline the state's response plan. The press conference will include media packets providing reporters with background information on CWD, a history of Michigan's surveillance efforts, and other materials as deemed needed or appropriate.

7. In the days following the announcement, public interest (and media attention) will be at peak levels. The PIOs for both agencies will coordinate efforts to have agency directors/designees engaged in public appearances or interviews in television and radio programs, as well as ensuring availabilities for print reporters and coordinating articles in stakeholder/trade publications to discuss the state's actions. Continual public communication will maximize public and media understanding of the situation.

8. Within 10 business days of the initial confirmation announcement, each agency will reactivate the communication teams employed in the surveillance plan to continue working as needed with local constituencies, facilitating communications, answering questions, and providing updates on Michigan's progress.

9. Each agency's press office will collect and analyze news stories to help determine the effectiveness, and modify as needed, the communication and outreach efforts. News and feature stories, as well as editorials and letters to the editor, will help indicate public awareness and understanding.

MICHIGAN DEPARTMENT OF AGRICULTURE AND RURAL DEVELOPMENT

Signed Date

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Keigh Creagh, Director Date

MICHIGAN DEPARTMENT OF NATURAL RESOURCES

Signed Date

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Rodney Stokes, Director Date

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Appendix A: Concise review of the scientific literature on CWD since the 2002 Plan

***Pathology/physiology*:** CWD as a disease process is now well described[5-39] and summarized in multiple reviews[40-68], pertaining both to free-ranging and captive cervids. The overwhelming preponderance of evidence shows that CWD is a transmissible spongiform encephalopathy (TSE), caused by a prion[5,6,10-17,24-27,31,35-40,42,45,46,51,55,57,68, among others]. Once subject to reasonable doubt, the prion theory is now essentially proven[67]. As one recent reviewer noted "the impressive recent progress … has removed all doubts about the prion hypothesis. …These findings have proven beyond any doubt that the prion hypothesis is indeed correct"[67]. The most plausible evidence to date suggests CWD probably originated when cervids became infected with some form of the domestic sheep disease scrapie[69-71].

Development of transgenic rodents (lab animals given the genetic susceptibility of other animals) has made major advances possible[10,17,72-80]. Sensitive and specific diagnostic tests are available[81-84], including tests for live deer and elk[85-92] which should soon facilitate test/cull programs and practical screening for captive cervids prior to interstate transit. Blood tests[93] and vaccines[28] for CWD are under development.

Two non-prion theories of CWD, one involving the *Spiroplasma* bacteria[94-98] and the other trace minerals[99-102] have been largely disproved. All the *Spiroplasma* studies have been carried out by only one laboratory, and independent researchers have been unable to replicate their findings[103,104]. According to a recent review "Based on the available data, the idea that prions consist of viruses or any other type of conventional microorganism is simply untenable"[67]. Research to date on trace minerals suggests that some like copper and manganese may affect susceptibility to CWD, but are incapable of causing the disease without infectious prions[99,100,105]. The most recent study suggests mineral alterations are more likely to be a result of CWD infection, not its cause[106].

***Epidemiology*:** With the exception of Korea, where it was imported by captive cervids[107,108], CWD has thus far only been found in North America, despite surveillance elsewhere[109-112]. In addition to the species (and subspecies) known to be susceptible in 2002 (white-tailed deer, mule deer and elk), moose (*Alces alces shirasi*)[113,114] and red deer (*Cervus elaphus elaphus*)[115,116] have been infected, and caribou (*Rangifer tarandus*) appear to be genetically susceptible[117], although no cases in free-ranging caribou[118] or red deer[110-112] have yet been found. Fallow deer (*Dama dama*) are not susceptible[119,120]. After limited surveillance, no cases have yet been found in roe deer (*Capreolus capreolus*)[110-112], Sika deer (*Cervus Nippon*)[109], or chamois (*Rupicapro rupicapro*)[112]. Some evidence suggests deer are more likely to transmit CWD than elk[29], and where both species occur over the same infected range, prevalence in deer is generally higher[63,121]. Within susceptible species, genetic variations mediate susceptibility, but thus far only in the sense that they affect the rapidity of disease progression, but not the inevitability of developing CWD[19,76,92,122-130]. Among non-cervids, cattle[18,20,21,131,132] and sheep[22]

are only susceptible experimentally, not under realistic exposure conditions[133]. Common furbearers that scavenge carcasses of infected cervids are thus far not susceptible[134-138]. Voles[139] and ferrets[140] are susceptible under experimental conditions only. Passage of the prion through one species can[8,141], but doesn't always[33], change its infectivity for other species. Such passage may mediate adaptation of distinct strains[30,33,139,140], and explain how prion diseases established in one species develop into new prion diseases in other species[55,67].

In infected cervids, CWD prions are present in blood[26,27], and so presumably in most perfused tissues. This has led to speculation that transmission by insect vectors might be possible[142], although there is no evidence thus far that it occurs. Saliva[16,17,26,27], aerosolized respiratory secretions[10,143], feces[79], urine[14,16,17], muscle[5,9,144], and antler velvet[6], but probably not semen or embryos[145], contain prions and are infectious. Infected cervids are infectious for susceptible animals months before they become symptomatic themselves[26,27,146].

Outbreaks of CWD in captive cervids[121,147-152], zoos[153], and free-ranging cervids in a variety of habitats[2,154-160] have been described in detail. Taken together, evidence suggests maintenance is by sustained horizontal (deer-to-deer) transmission[150-152,161], with exposures from prion-contaminated environments playing a critical role[121,149,162-164]. In the wild, prevalence increases with age, and is higher in males[154,155,157,165], and can dramatically lower survival[3], with mature bucks showing the highest rates of CWD-associated mortality[3,154]. Infected does raise fewer fawns to weaning, but the primary constraint on population growth rate is higher mortality rather than decreased recruitment[166]. Infected animals are more likely to be hit by vehicles[167], and to be killed by predators[3]. Increasing carrying capacity may increase the likelihood of disease establishment and persistence[162,168]. Despite initial arguments about the appropriateness of models[169], model predictions of high prevalence, growing rates of infection and substantial decreases in population abundance[158] are now a reality evident in some infected areas[3,157,165]. Transmission has both density and frequency dependent characteristics[170,171], which may suggest transmission increases with cervid density prior to substantial environmental contamination (i.e. establishment), and gradually becomes less density dependent thereafter[163,165]. Some models suggest CWD prevalence and the severity of population declines are driven by the amount of time prions remain infectious in the environment[162,163]. The high frequency of contacts in deer family groups promotes local maintenance of CWD[159,161,172-176]. Both migratory[177,178] and dispersal[160,161,175,179] movements are likely responsible for geographic spread depending upon species and habitat. Land use by humans also plays a role[168,171,180]. Risk of spread gradually diminishes with distance[156,174], and is affected by geographic barriers (e.g. large rivers, highways) . Areas where cervids congregate repeatedly have a higher risk of becoming CWD contaminated[159,172,177,181]. Risk of transmission between captive and free-ranging cervids by contact across intact fences appears greater for elk than deer[182,183].

Prions are very persistent in soils[58,121] (e.g., scrapie prions persist for at least 16 years;[184]). They bind strongly to soils[53,185,186], to clays stronger than to sand[187,188], and retain[189], and greatly enhance[190], their infectivity. This may explain the spread of some TSEs in spite of the low levels shed into the environment by infected animals. Once bound to soil[164] or sewage sludge[191], there is minimal prion desorption into water[192]. Where manganese oxides are rich in soils, they may help degrade prions[193]. Mapped at a landscape scale, clay content of soils in CWD-infected areas is an important predictor of infection odds[194].

Although there has been considerable concern that CWD might infect humans[46,195-199], ongoing epidemiological studies have not identified any human cases to date[195-198,200-202]. Laboratory studies in transgenic mice[74,203] and those non-human primates evolutionarily closer to humans[204] thus far suggest humans are not susceptible. Nevertheless, public health officials continue to assemble human exposure data[199,200,205,206]. A new human prion has been created from CWD in one laboratory[141], but whether those results could also occur naturally is unclear. Because different strains of CWD exist[207], and others may develop, unless all strains are tested, the possibility of human infections cannot be completely excluded. However, to date, there is no evidence CWD as it currently exists can infect humans, and considerable evidence that infection is extremely unlikely, if possible at all.

***Human dimensions*:** Early criticism of CWD management pointed out the need for human dimensions research and public engagement[208]. Hunter's perceptions of shared goals and values with state wildlife agencies positively influenced their trust in those agencies to manage CWD, but had little effect on their perceptions about health risks from CWD[209]. As perceived health risks and CWD prevalence increase, non-resident hunters were increasingly likely to hunt elsewhere, while residents were likely to quit hunting[210]. Newcomers to hunting were the most likely to quit, a prospect called "catastrophic" for hunter recruitment[210]. Fifty-two percent of Wisconsin hunters who did not hunt in 2002 quit because of CWD, and they were less likely to believe and trust the state wildlife agency's information on CWD[211]. A more recent study[212] found CWD prevalence was the strongest predictor of quitting hunting, followed by human health risks, estimating that 64% of hunters would quit in the worst-case scenario. In Wisconsin, most non-hunting landowners in the CWD eradication zone were neutral or unconcerned with CWD and its management, while most hunters were concerned, mostly with the effect of CWD on deer hunting and the safety of eating venison[213]. Hunting efficiency, number of deer seen by hunters, willingness to harvest antlerless deer, and desire for venison predicted deer harvest levels better than time afield[214]. A Michigan study found the public particularly supportive of lethal management of wildlife to control diseases[215]. A statistical index has been developed to assess the potential for conflict between hunter attitudes and potential agency CWD management actions[216], as has a choice model of Michigan residents' preferences for deer management where trade offs are necessary[217]. Agency attempts to provide CWD information to the public by a variety of methods have had limited success[4,218]. A critique of CWD information presented on government agency web sites also exists[219].

***Management*:** Surveillance for CWD using hunter harvested deer alone often has a low probability of detecting the disease[220] and may give biased results[221,222]. Alternatives have been developed[222,223]. By the time symptomatic free-ranging deer are detected, prevalence is typically >1%[2,158].

Higher CWD prevalence has been associated with human land development, which may be related to supplemental feeding, smaller home ranges, refuge from hunting and/or predators, concentrating deer on fewer patches of good habitat[90,168] or overlapping space use[224]. A test and cull strategy for managing CWD in urban deer has been evaluated[91]. Transmission[159] and geographic spread of CWD is influenced by landscape features such as rivers[225], mountains and roads[226] percent forest cover[227,228] and clay content of soils[194]. Support exists for targeting buck fawns and yearling bucks because of the risk of CWD spread from dispersal of yearling bucks[160,175] in riparian habitats[229], and entire family groups where one or more of the female members are CWD-positive[161,174,175]. The potential for spread via migration may be greater in northern latitudes[178]. The potential for spread via atypical long distance movements, although low[160], has also been pointed out[230]. Thus far, although recommended for high prevalence areas[156,170], culling deer has not been shown to significantly decrease prevalence where CWD is known to be established[231]. However, density reductions (due to culling, or otherwise) may successfully control disease spread to uninfected areas[165]. Non-hunted deer populations sustain high levels of infection, generating substantial risk of disease spread[170]. Once established, CWD outbreaks (and the necessity and costs of their management) can be expected to last for decades[170].

Evidence suggests mountain lions preferentially prey upon CWD-infected deer[3,232], and that even intense predation is insufficient to limit the spread and persistence of the disease[3]. As deer die from CWD, local imbalances in predator/prey ecology may be likely, with effects on other important species[3]. Thus far, infected elk appear less prone to such predation, although CWD remains a leading cause of death[233]. Selective predation by wolves may suppress CWD establishment or limit prevalence more effectively than hunting or culling, while reducing deer population size more modestly[234].

In Wisconsin, research supports the use of earn-a-buck (EAB) as a strategy for increasing deer harvest. The use of EAB coupled with more days of hunting opportunity was 56-88% more effective at increasing antlerless harvest than extended opportunity alone[235], but EAB was disliked by many hunters[235] and few hunters shoot multiple antlerless deer in order to get multiple buck tags[214]. Hunter harvest was positively related to deer density, landowner requests for shooting permits, and proximity to high CWD prevalence areas, but unaffected by hunter boycotts of deer reduction strategies[236]. Monetary incentives to reward hunters for shooting more deer appear largely ineffective[214]. Hunter harvest decisions depend most heavily on personal attitudes and are relatively unaffected by agency attempts to increase them. “Managers may be best served to manage the segment of hunters

most willing to harvest deer rather than taking a broad approach of providing a longer season as a means of increasing effort"[214].

Management practices that increase carrying capacity may cause CWD to persist and even destabilize populations, especially where prions persist in the environment[162]. Contact, and so the potential for CWD spread, between doe groups occurs mainly during feeding[172], and is intensified by supplemental feeding compared to natural foraging behavior[237]. Supplemental feeding (and likely baiting as well) of deer by humans contributes to spread of CWD, causes habitat destruction near feeders, crowding, fighting and injuries, and starvation due to compensatory increases in population above carrying capacity[238]. Alternative restrictions on the quantity of supplemental feed do not mitigate the potential for CWD transmission[237]. Where supplemental feeding has been critically studied, "none of the feeding strategies evaluated substantially reduced the potential risk for disease transmission and banning supplemental feeding to reduce transmission is warranted"[237].

The potential for management of CWD via vaccines[28] and prion-inactivating chemicals[193,239] is an area of active research, but still in its infancy. The unique biology of prions compared to other infectious agents makes progress in these areas slow and difficult.

Management of CWD spread via movement of captive cervids is comparatively straightforward, consisting of rigorous testing (which will be facilitated by the recent availability of live animal tests:[86,90,92]), enforcement of movement restrictions on commercial animals, and protocols for screening of free-ranging animals prior to translocation[240].

References

1. Smith, C.A., *The role of state wildlife professionals under the public trust doctrine.* Journal of Wildlife Management. **75**(7): p. 1539-1543.
2. Joly, D.O., et al., *Chronic wasting disease in free-ranging Wisconsin white-tailed deer.* Emerging Infectious Diseases, 2003. **9**(5): p. 599-601.
3. Miller, M.W., et al., *Lions and prions and deer demise.* PLoS ONE, 2008. **3**(12): p. e4019.
4. Brown, T.L., et al., *Hunters' and other citizens' reactions to discovery of CWD in central New York.* Human Dimensions of Wildlife, 2006. **11**(3): p. 203-214.
5. Angers, R.C., et al., *Prions in skeletal muscles of deer with chronic wasting disease.* Science, 2006. **311**(5764): p. 1117-1117.
6. Angers, R.C., et al., *Chronic Wasting Disease prions in elk antler velvet.* Emerging Infectious Diseases, 2009. **15**(5): p. 696-703.
7. Ball, K., *Chronic wasting disease in a Rocky Mountain elk.* Canadian Veterinary Journal-Revue Veterinaire Canadienne, 2002. **43**(11): p. 880-882.
8. Bartz, J.C., et al., *The host range of chronic wasting disease is altered on passage in ferrets.* Virology, 1998. **251**(2): p. 297-301.
9. Daus, M.L., et al., *Presence and seeding activity of pathological prion protein (PrP(TSE)) in skeletal muscles of white-tailed deer infected with Chronic Wasting Disease.* Plos One, 2011. **6**(4): p. 7.
10. Denkers, N.D., et al., *Aerosol and nasal transmission of chronic wasting disease in cervidized mice.* Journal of General Virology, 2010. **91**: p. 1651-1658.
11. Denkers, N.D., G.C. Telling, and E.A. Hoover, *Minor oral lesions facilitate transmission of Chronic Wasting Disease.* Journal of Virology, 2011. **85**(3): p. 1396-1399.

12. Fox, K.A., et al., *Patterns of Prp(CWD) accumulation during the course of chronic wasting disease infection in orally inoculated mule deer (Odocoileus hemionus).* Journal of General Virology, 2006. **87**: p. 3451-3461.
13. Goldmann, W., *PrP genetics in ruminant transmissible spongiform encephalopathies.* Veterinary Research, 2008. **39**(4).
14. Gonzalez-Romero, D., et al., *Detection of infectious prions in urine.* Febs Letters, 2008. **582**(21-22): p. 3161-3166.
15. Green, K.M., et al., *The elk PRNP codon 132 polymorphism controls cervid and scrapie prion propagation.* Journal of General Virology, 2008. **89**: p. 598-608.
16. Haley, N.J., et al., *Detection of CWD prions in salivary and urinary tissues of deer: Potential mechanisms of pathogenesis and prion shedding.* Prion, 2010. **4**(3): p. 150-150.
17. Haley, N.J., et al., *Detection of CWD prions in urine and saliva of deer by transgenic mouse bioassay.* Plos One, 2009. **4**(3).
18. Hamir, A.N., et al., *Preliminary findings on the experimental transmission of chronic wasting disease agent of mule deer to cattle.* Journal of Veterinary Diagnostic Investigation, 2001. **13**(1): p. 91-96.
19. Hamir, A.N., et al., *Preliminary observations of genetic susceptibility of elk (Cervus elaphus nelsoni) to chronic wasting disease by experimental oral inoculation.* Journal of Veterinary Diagnostic Investigation, 2006. **18**(1): p. 110-114.
20. Hamir, A.N., et al., *Experimental interspecies transmission studies of the transmissible spongiform encephalopathies to cattle: comparison to bovine spongiform encephalopathy in cattle.* Journal of Veterinary Diagnostic Investigation, 2011. **23**(3): p. 407-420.
21. Hamir, A.N., et al., *Experimental transmission of chronic wasting disease agent from mule deer to cattle by the intracerebral route.* Journal of Veterinary Diagnostic Investigation, 2005. **17**(3): p. 276-281.
22. Hamir, A.N., et al., *Transmission of chronic wasting disease of mule deer to Suffolk sheep following intracerebral inoculation.* Journal of Veterinary Diagnostic Investigation, 2006. **18**(6): p. 558-565.
23. Hamir, A.N., J.M. Miller, and R.C. Cutlip, *Failure to detect prion protein (PrPres) by immunohistochemistry in striated muscle tissues of animals experimentally inoculated with agents of transmissible spongiform encephalopathy.* Veterinary Pathology, 2004. **41**(1): p. 78-81.
24. Liberski, P.P., et al., *Deposition patterns of disease-associated prion protein in captive mule deer brains with chronic wasting disease.* Acta Neuropathologica, 2001. **102**(5): p. 496-500.
25. Mathiason, C.K., et al., *B Cells and Platelets Harbor Prion Infectivity in the Blood of Deer Infected with Chronic Wasting Disease.* Journal of Virology, 2010. **84**(10): p. 5097-5107.
26. Mathiason, C.K., et al., *Infectious prions in pre-clinical deer and transmission of Chronic Wasting Disease solely by environmental exposure.* Plos One, 2009. **4**(6): p. 9.
27. Mathiason, C.K., et al., *Infectious prions in the saliva and blood of deer with chronic wasting disease.* Science, 2006. **314**(5796): p. 133-136.
28. Pilon, J., et al., *Anti-prion activity generated by a novel vaccine formulation.* Neuroscience Letters, 2007. **429**(2-3): p. 161-164.
29. Race, B.L., et al., *Levels of abnormal prion protein in deer and elk with chronic wasting disease.* Emerging Infectious Diseases, 2007. **13**(6): p. 824-830.
30. Race, R.E., et al., *Comparison of abnormal prion protein glycoform patterns from transmissible spongiform encephalopathy agent-infected deer, elk, sheep, and cattle.* Journal of Virology, 2002. **76**(23): p. 12365-12368.
31. Safar, J.G., et al., *Transmission and detection of prions in feces.* Journal of Infectious Diseases, 2008. **198**(1): p. 81-89.
32. Sigurdson, C.J., et al., *PrPCWD lymphoid cell targets in early and advanced chronic wasting disease of mule deer.* Journal of General Virology, 2002. **83**: p. 2617-2628.
33. Sigurdson, C.J., et al., *Strain fidelity of chronic wasting disease upon murine adaptation.* Journal of Virology, 2006. **80**(24): p. 12303-12311.
34. Sigurdson, C.J., et al., *PrPCWD in the myenteric plexus, vagosympathetic trunk and endocrine glands of deer with chronic wasting disease.* Journal of General Virology, 2001. **82**: p. 2327-2334.
35. Slate, J., *Molecular evolution of the sheep prion protein gene.* Proceedings of the Royal Society B-Biological Sciences, 2005. **272**(1579): p. 2371-2377.
36. Spraker, T.R., et al., *Variable patterns of distribution of Prp(CWD) in the obex and cranial lymphoid tissues of Rocky Mountain elk (Cervus elaphus nelsoni) with subclinical chronic wasting disease.* Veterinary Record, 2004. **155**(10): p. 295-302.
37. Spraker, T.R., et al., *Distribution of protease-resistant prion protein and spongiform encephalopathy in free-ranging mule deer (Odocoileus hemionus) with chronic wasting disease.* Veterinary Pathology, 2002. **39**(5): p. 546-556.
38. Spraker, T.R., et al., *Comparison of histological lesions and immunohistochemical staining of proteinase-resistant prion protein in a naturally occurring spongiform encephalopathy of free-ranging mule deer (Odocoileus hemionus) with those of chronic wasting disease of captive mule deer.* Veterinary Pathology, 2002. **39**(1): p. 110-119.

39. Xie, Z.L., et al., *Chronic wasting disease of elk and deer and Creutzfeldt-Jakob disease - Comparative analysis of the scrapie prion protein.* Journal of Biological Chemistry, 2006. **281**(7): p. 4199-4206.
40. Beekes, M. and P.A. McBride, *The spread of prions through the body in naturally acquired transmissible spongiform encephalopathies.* Febs Journal, 2007. **274**(3): p. 588-605.
41. Beiglbock, C., *Chronic Wasting Disease (CWD) in cervids in North America - a review.* Wiener Tierarztliche Monatsschrift, 2001. **88**(6): p. 147-152.
42. Collinge, J., *Prion diseases of humans and animals: Their causes and molecular basis.* Annual Review of Neuroscience, 2001. **24**: p. 519-550.
43. Conner, M.M., et al., *Infectious disease in cervids of north America - Data, models, and management challenges.* Annals of the New York Academy of Science, 2008. **1134**: p. 146-172.
44. Dagleish, M.P., *Chronic wasting disease in deer and elk.* Veterinary Record, 2004. **155**(23): p. 750-750.
45. Doherr, M.G., *Brief review on the epidemiology of transmissible spongiform encephalopathies (TSE).* Vaccine, 2007. **25**(30): p. 5619-5624.
46. Dormont, D., *Prion diseases: pathogenesis and public health concerns.* Febs Letters, 2002. **529**(1): p. 17-21.
47. Haigh, J.C., C. Mackintosh, and F. Griffin, *Viral, parasitic and prion diseases of farmed deer and bison.* Revue Scientifique Et Technique De L Office International Des Epizooties, 2002. **21**(2): p. 219-248.
48. Kahn, S., et al., *Chronic wasting disease in Canada: Part 1.* Canadian Veterinary Journal-Revue Veterinaire Canadienne, 2004. **45**(5): p. 397-404.
49. Kellar, J.A. and V.W. Lees, *Risk management of the transmissible spongiform encephalopathies in North America.* Revue Scientifique Et Technique De L Office International Des Epizooties, 2003. **22**(1): p. 201-225.
50. Miller, M.W. and E.S. Williams, *Chronic wasting disease of cervids*. Current Topics in Microbiology and Immunology, 2004. **284**: p. 193-214.
51. Ryou, C., *Prions and prion diseases: Fundamentals and mechanistic details.* Journal of Microbiology and Biotechnology, 2007. **17**(7): p. 1059-1070.
52. Salman, M.D., *Chronic wasting disease in deer and elk: Scientific facts and findings.* Journal of Veterinary Medical Science, 2003. **65**(7): p. 761-768.
53. Saunders, S.E., S.L. Bartelt-Hunt, and J.C. Bartz, *Prions in the environment: Occurrence, fate and mitigation.* Prion, 2008. **2**(4): p. 162-169.
54. Sejvar, J.J., L.B. Schonberger, and E.D. Belay, *Transmissible spongiform encephalopathies.* Javma-Journal of the American Veterinary Medical Association, 2008. **233**(11): p. 1705-1712.
55. Sigurdson, C.J., *A prion disease of cervids: Chronic wasting disease.* Veterinary Research, 2008. **39**(4).
56. Sigurdson, C.J. and M.W. Miller, *Other animal prion diseases.* British Medical Bulletin, 2003. **66**: p. 199-212.
57. Silveira, J.R., B. Caughey, and G.S. Baron, *Prion protein and the molecular features of transmissible spongiform encephalopathy agents*. Current Topics in Microbiology and Immunology, 2004. **284**: p. 1-50.
58. Smith, C.B., C.J. Booth, and J.A. Pedersen, *Fate of Prions in Soil: A Review.* Journal of Environmental Quality, 2011. **40**(2): p. 449-461.
59. Travis, D. and M. Miller, *A short review of transmissible spongiform encephalopathies, and guidelines for managing risks associated with chronic wasting disease in captive cervids in zoos.* Journal of Zoo and Wildlife Medicine, 2003. **34**(2): p. 125-133.
60. Williams, E. and M.W. Miller, *Chronic wasting disease in cervids.* Brain Pathology, 2000. **10**(4): p. 608-608.
61. Williams, E.S., *The transmissible spongiform encephalopathies: disease risks for North America.* Veterinary Clinics of North America-Food Animal Practice, 2002. **18**(3): p. 461-+.
62. Williams, E.S., *Scrapie and chronic wasting disease.* Clinics in Laboratory Medicine, 2003. **23**(1): p. 139-+.
63. Williams, E.S., *Chronic wasting disease.* Veterinary Pathology, 2005. **42**(5): p. 530-549.
64. Williams, E.S. and M.W. Miller, *Chronic wasting disease in deer and elk in North America.* Revue Scientifique Et Technique De L Office International Des Epizooties, 2002. **21**(2): p. 305-316.
65. Williams, E.S. and M.W. Miller, *Transmissible spongiform encephalopathies in non-domestic animals: origin, transmission and risk factors.* Revue Scientifique Et Technique De L Office International Des Epizooties, 2003. **22**(1): p. 145-156.
66. Williams, E.S., et al., *Chronic wasting disease of deer and elk: A review with recommendations for management.* Journal of Wildlife Management, 2002. **66**(3): p. 551-563.
67. Soto, C., *Prion hypothesis: the end of the controversy?* Trends in Biochemical Sciences, 2011. **36**(3): p. 151-158.
68. Tyler, J.W. and J.R. Middleton, *Transmissible spongiform encephalopathies in ruminants.* Veterinary Clinics of North America-Food Animal Practice, 2004. **20**(2): p. 303-+.
69. Greenlee, J.J., J.D. Smith, and R.A. Kunkle, *White-tailed deer are susceptible to the agent of sheep scrapie by intracerebral inoculation.* Veterinary Research, 2011. **42**: e-resource #10.1186/1297-9716-42-107.

70. Hamir, A.N., et al., *Transmission of sheep scrapie to elk (Cervus elaphus nelsoni) by intracerebral inoculation: final outcome of the experiment.* Journal of Veterinary Diagnostic Investigation, 2004. **16**(4): p. 316-321.
71. Hamir, A.N., et al., *Preliminary observations on the experimental transmission of scrapie to elk (Cervus elaphus nelsoni) by intracerebral inoculation.* Veterinary Pathology, 2003. **40**(1): p. 81-85.
72. Browning, S.R., et al., *Transmission of prions from mule deer and elk with chronic wasting disease to transgenic mice expressing cervid PrP.* Journal of Virology, 2004. **78**(23): p. 13345-13350.
73. Groschup, M.H. and A. Buschmann, *Rodent models for prion diseases.* Veterinary Research, 2008. **39**(4): e-resource #10.1051/vetres:2008008.
74. Kong, Q.Z., et al., *Chronic wasting disease of elk: Transmissibility to humans examined by transgenic mouse models.* Journal of Neuroscience, 2005. **25**(35): p. 7944-7949.
75. LaFauci, G., et al., *Passage of chronic wasting disease prion into transgenic mice expressing Rocky Mountain elk (Cervus elaphus nelsoni) PrP^{C}.* Journal of General Virology, 2006. **87**: p. 3773-3780.
76. Meade-White, K., et al., *Resistance to chronic wasting disease in transgenic mice expressing a naturally occurring allelic variant of deer prion protein.* Journal of Virology, 2007. **81**(9): p. 4533-4539.
77. Sandberg, M.K., et al., *Chronic wasting disease prions are not transmissible to transgenic mice overexpressing human prion protein.* Journal of General Virology, 2010. **91**: p. 2651-2657.
78. Tamguney, G., et al., *Transmission of elk and deer prions to transgenic mice.* Journal of Virology, 2006. **80**(18): p. 9104-9114.
79. Safar, J.G., et al., *Measuring prions causing bovine spongiform encephalopathy or chronic wasting disease by immunoassays and transgenic mice.* Nature Biotechnology, 2002. **20**(11): p. 1147-1150.
80. Trifilo, M.J., et al., *Chronic wasting disease of deer and elk in transgenic mice: Oral transmission and pathobiology.* Virology, 2007. **365**(1): p. 136-143.
81. Miller, M.W. and E.S. Williams, *Detection of PrP^{CWD} in mule deer by immunohistochemistry of lymphoid tissues.* Veterinary Record, 2002. **151**(20): p. 610-612.
82. Hibler, C.P., et al., *Field validation and assessment of an enzyme-linked immunosorbent assay for detecting chronic wasting disease in mule deer (Odocoileus hemionus), white-tailed deer (Odocoileus virginianus), and Rocky Mountain elk (Cervus elaphus nelsoni).* Journal of Veterinary Diagnostic Investigation, 2003. **15**(4): p. 311-319.
83. Keane, D.P., et al., *Comparison of retropharyngeal lymph node and obex region of the brainstem in detection of chronic wasting disease in white-tailed deer (Odocoileus virginianus).* Journal of Veterinary Diagnostic Investigation, 2008. **20**(1): p. 58-60A.
84. Spraker, T.R., et al., *Validation of monoclonal antibody F99/97.6.1 for immunohistochemical staining of brain and tonsil in mule deer (Odocoileus hemionus) with chronic wasting disease.* Journal of Veterinary Diagnostic Investigation, 2002. **14**(1): p. 3-7.
85. Spraker, T.R., et al., *Detection of PrPCWD in postmortem rectal lymphoid tissues in Rocky Mountain elk (Cervus elaphus nelsoni) infected with chronic wasting disease.* Journal of Veterinary Diagnostic Investigation, 2006. **18**(6): p. 553-557.
86. Spraker, T.R., et al., *Antemortem detection of PrP(CWD) in preclinical, ranch-raised Rocky Mountain elk (Cervus elaphus nelsoni) by biopsy of the rectal mucosa.* Journal of Veterinary Diagnostic Investigation, 2009. **21**(1): p. 15-24.
87. Keane, D., et al., *Validation of use of rectoanal mucosa-associated lymphoid tissue for immunohistochemical diagnosis of Chronic Wasting Disease in white-tailed deer (Odocoileus virginianus).* Journal of Clinical Microbiology, 2009. **47**(5): p. 1412-1417.
88. Schuler, K.L., et al., *Tonsillar biopsy test for chronic wasting disease: Two sampling approaches in mule deer and white-tailed deer.* Journal of Wildlife Diseases, 2005. **41**(4): p. 820-824.
89. Wild, M.A., et al., *Preclinical diagnosis of chronic wasting disease in captive mule deer (Odocoileus hemionus) and white-tailed deer (Odocoileus virginianus) using tonsillar biopsy.* Journal of General Virology, 2002. **83**: p. 2629-2634.
90. Wolfe, L.L., et al., *Evaluation of antemortem sampling to estimate chronic wasting disease prevalence in free-ranging mule deer.* Journal of Wildlife Management, 2002. **66**(3): p. 564-573.
91. Wolfe, L.L., M.W. Miller, and E.S. Williams, *Feasibility of "test-and-cull" for managing chronic wasting disease in urban mule deer.* Wildlife Society Bulletin, 2004. **32**(2): p. 500-505.
92. Wolfe, L.L., et al., *PrPCWD in rectal lymphoid tissue of deer (Odocoileus spp.).* Journal of General Virology, 2007. **88**: p. 2078-2082.
93. Chang, B.G., et al., *Test for detection of disease-associated prion aggregate in the blood of infected but asymptomatic animals.* Clinical and Vaccine Immunology, 2007. **14**(1): p. 36-43.
94. Bastian, F.O., *Spiroplasma as a candidate agent for the transmissible spongiform encephalopathies.* Journal of Neuropathology and Experimental Neurology, 2005. **64**(10): p. 833-838.
95. Bastian, F.O., S. Dash, and R.F. Garry, *Linking chronic wasting disease to scrapie by comparison of Spiroplasma mirum ribosomal DNA sequences.* Experimental and Molecular Pathology, 2004. **77**(1): p. 49-56.

96. Bastian, F.O. and C.D. Fermin, *Slow virus disease: Deciphering conflicting data on the transmissible spongiform encephalopathies (TSE) also called prion diseases.* Microscopy Research and Technique, 2005. **68**(3-4): p. 239-246.
97. Bastian, F.O., et al., *Experimental Spiroplasma mirum infection in deer: An animal model for chronic wasting disease.* Brain Pathology, 2006. **16**: p. S15-S15.
98. Bastian, F.O., et al., *Spiroplasma spp. from transmissible spongiform encephalopathy brains or ticks induce spongiform encephalopathy in ruminants.* Journal of Medical Microbiology, 2007. **56**(9): p. 1235-1242.
99. Flood, P., *The influence of trace minerals on susceptibility to chronic wasting disease (CWD).* Final report submitted to the Saskatchewan Department of Agriculture and Food, Agricultural Development Fund, 25 November 2003, Project 20010027, RE24AD, 2003. Western College of Veterinary Medicine, Univ. of Saskatchewan: Saskatoon, SK. 15 pp.
100. Leach, S.P., M.D. Salman, and D. Hamar, *Trace elements and prion diseases: a review of the interactions of copper, manganese and zinc with the prion protein.* Animal Health Research Reviews, 2007. **7**(1-2): p. 97-105.
101. McBride, M.B., *Trace metals and sulfur in soils and forage of a chronic wasting disease locus.* Environmental Chemistry, 2007. **4**(2): p. 134-139.
102. Purdey, M., *Elevated silver, barium and strontium in antlers, vegetation and soils sourced from CWD cluster areas: Do Ag/Ba/Sr piezoelectric crystals represent the transmissible pathogenic agent in TSEs?* Medical Hypotheses, 2004. **63**(2): p. 211-225.
103. Alexeeva, I., et al., *Absence of Spiroplasma or other bacterial 16S rRNA genes in brain tissue of hamsters with scrapie.* Journal of Clinical Microbiology, 2006. **44**(1): p. 91-97.
104. Hamir, A.N., et al., *Experimental inoculation of raccoons (Procyon lotor) with Spiroplasma mirum and transmissible mink encephalopathy (TME).* Canadian Journal of Veterinary Research-Revue Canadienne De Recherche Veterinaire, 2011. **75**(1): p. 18-24.
105. Millhauser, G.L., *Copper and the prion protein: Methods, structures, function, and disease.* Annual Review of Physical Chemistry, 2007. **58**: p. 299-320.
106. Wolfe, L.L., et al., *Select tissue mineral concentrations and Chronic Wasting Disease status in mule deer from north-central Colorado.* Journal of Wildlife Diseases, 2010. **46**(3): p. 1029-1034.
107. Kim, T.Y., et al., *Additional cases of chronic wasting disease in imported deer in Korea.* Journal of Veterinary Medical Science, 2005. **67**(8): p. 753-759.
108. Sohn, H.J., et al., *A case of chronic wasting disease in an elk imported to Korea from Canada.* Journal of Veterinary Medical Science, 2002. **64**(9): p. 855-858.
109. Kataoka, N., et al., *Surveillance of chronic wasting disease in sika deer, Cervus nippon, from Tokachi district in Hokkaido.* Journal of Veterinary Medical Science, 2005. **67**(3): p. 349-351.
110. Roels, S., et al., *First results of chronic wasting disease (CWD) surveillance in the South-Eastern part of Belgium.* Veterinary Quarterly, 2005. **27**(3): p. 98-104.
111. Schettler, E., et al., *Surveillance for prion disease in Cervids, Germany.* Emerging Infectious Diseases, 2006. **12**(2): p. 319-322.
112. Schwaiger, K., et al., *Survey on transmissible spongiform encephalopathies in roe deer (Capreolus capreolus), red deer (Cervus elaphus) and chamois (Rupicapro rupicapro) in Bavaria.* Berliner Und Munchener Tierarztliche Wochenschrift, 2004. **117**(1-2): p. 24-29.
113. Baeten, L.A., et al., *A natural case of chronic wasting disease in a free-ranging moose (Alces alces shirasi).* Journal of Wildlife Diseases, 2007. **43**(2): p. 309-314.
114. Kreeger, T.J., et al., *Oral transmission of chronic wasting disease in captive Shira's moose.* Journal of Wildlife Diseases, 2006. **42**(3): p. 640-645.
115. Balachandran, A., et al., *Experimental oral transmission of chronic wasting disease to red deer (Cervus elaphus elaphus): Early detection and late stage distribution of protease-resistant prion protein.* Canadian Veterinary Journal-Revue Veterinaire Canadienne, 2010. **51**(2): p. 169-178.
116. Dagleish, M.P., et al., *Experimental transmission of bovine spongiform encephalopathy to European red deer (Cervus elaphus elaphus).* BMC Veterinary Research, 2008. **4**.
117. Happ, G.M., et al., *Prion protein genes in caribou from Alaska.* Journal of Wildlife Diseases, 2007. **43**(2): p. 224-228.
118. Lapointe, J.M., et al., *Screening for chronic wasting disease in caribou in northern Quebec.* Canadian Veterinary Journal-Revue Veterinaire Canadienne, 2002. **43**(11): p. 886-887.
119. Hamir, A.N., et al., *Preliminary observations on the experimental transmission of chronic wasting disease (CWD) from elk and white-tailed deer to fallow deer.* Journal of Comparative Pathology, 2008. **138**(2-3): p. 121-130.
120. Rhyan, J.C., et al., *Failure of fallow deer (Dama dama) to develop Chronic Wasting Disease when exposed to a contaminated environment and infected mule deer (Odocoileus hemionus).* Journal of Wildlife Diseases, 2011. **47**(3): p. 739-744.
121. Miller, M.W., et al., *Environmental sources of prion transmission in mule deer.* Emerging Infectious Diseases, 2004. **10**(6): p. 1003-1006.

122. Huson, H.J. and G.M. Happ, *Polymorphisms of the prion protein gene (PRNP) in Alaskan moose (Alces alces gigas).* Animal Genetics, 2006. **37**(4): p. 425-426.
123. Jewell, J.E., et al., *Low frequency of PrP genotype 225SF among free-ranging mule deer (Odocoileus hemionus) with chronic wasting disease.* Journal of General Virology, 2005. **86**: p. 2127-2134.
124. Johnson, C., et al., *Prion protein gene heterogeneity in free-ranging white-tailed deer within the chronic wasting disease affected region of Wisconsin.* Journal of Wildlife Diseases, 2003. **39**(3): p. 576-581.
125. Johnson, C., et al., *Prion protein polymorphisms in white-tailed deer influence susceptibility to chronic wasting disease.* Journal of General Virology, 2006. **87**: p. 2109-2114.
126. O'Rourke, K.I., et al., *Polymorphisms in the prion precursor functional gene but not the pseudogene are associated with susceptibility to chronic wasting disease in white-tailed deer.* Journal of General Virology, 2004. **85**: p. 1339-1346.
127. O'Rourke, K.I., et al., *Elk with a long incubation prion disease phenotype have a unique PrPd profile.* Neuroreport, 2007. **18**(18): p. 1935-1938.
128. Richt, J.r.A. and S.M. Hall, *BSE case associated with prion protein gene mutation.* PLoS Pathogens, 2008. **4**(9): e1000156.
129. Wilson, G.A., et al., *Polymorphisms at the PRNP Gene Influence Susceptibility to Chronic Wasting Disease in Two Species of Deer (Odocoileus Spp.) in Western Canada.* Journal of Toxicology and Environmental Health-Part A-Current Issues, 2009. **72**(17-18): p. 1025-1029.
130. Perucchini, M., et al., *PrP genotypes of free-ranging wapiti (Cervus elaphus nelsoni) with chronic wasting disease.* Journal of General Virology, 2008. **89**: p. 1324-1328.
131. Hamir, A.N., et al., *Susceptibility of cattle to first-passage intracerebral inoculation with chronic wasting disease agent from white-tailed deer.* Veterinary Pathology, 2007. **44**(4): p. 487-493.
132. Hamir, A.N., et al., *Experimental second passage of chronic wasting disease (CWDmule (deer)) agent to cattle.* Journal of Comparative Pathology, 2006. **134**(1): p. 63-69.
133. Gould, D.H., et al., *Survey of cattle in northeast Colorado for evidence of chronic wasting disease: geographical and high-risk targeted sample.* Journal of Veterinary Diagnostic Investigation, 2003. **15**(3): p. 274-277.
134. Harrington, R.D., et al., *A species barrier limits transmission of chronic wasting disease to mink (Mustela vison).* Journal of General Virology, 2008. **89**: p. 1086-1096.
135. Jennelle, C.S., et al., *Deer carcass decomposition and potential scavenger exposure to Chronic Wasting Disease.* Journal of Wildlife Management, 2009. **73**(5): p. 655-662.
136. Jennelle, C.S., et al., *Surveillance for transmissible spongiform encephalopathy in scavengers of white-tailed deer carcasses in the Chronic Wasting Disease area of Wisconsin.* Journal of Toxicology and Environmental Health-Part A-Current Issues, 2009. **72**(17-18): p. 1018-1024.
137. Hamir, A.N., et al., *Age-related lesions in laboratory-confined raccoons (Procyon lotor) inoculated with the agent of chronic wasting disease of mule deer.* Journal of Veterinary Diagnostic Investigation, 2007. **19**(6): p. 680-686.
138. Hamir, A.N., et al., *Experimental inoculation of scrapie and chronic wasting disease agents in raccoons (Procyon lotor).* Veterinary Record, 2003. **153**(4): p. 121-123.
139. Heisey, D.M., et al., *Chronic Wasting Disease (CWD) susceptibility of several North American rodents that are sympatric with cervid CWD epidemics.* Journal of Virology, 2011. **84**(1): p. 210-215.
140. Sigurdson, C.J., et al., *Experimental chronic wasting disease (CWD) in the ferret.* Journal of Comparative Pathology, 2008. **138**(4): p. 189-196.
141. Barria, M.A., et al., *Generation of a new form of human PrP(Sc) in vitro by interspecies transmission from cervid prions.* Journal of Biological Chemistry, 2011. **286**(9): p. 7490-7495.
142. Lupi, O., *Risk analysis of ectoparasites acting as vectors for chronic wasting disease.* Medical Hypotheses, 2005. **65**(1): p. 47-54.
143. Haybaeck, J., et al., *Aerosols transmit prions to immunocompetent and immunodeficient mice.* Plos Pathogens, 2011. **7**(1): p. 19.
144. Jewell, J.E., et al., *Prion protein in cardiac muscle of elk (Cervus elaphus nelsoni) and white-tailed deer (Odocoileus virginianus) infected with chronic wasting disease.* Journal of General Virology, 2006. **87**: p. 3443-3450.
145. Wrathall, A.E., et al., *Risks of transmitting ruminant spongiform encephalopathies (prion diseases) by semen and embryo transfer techniques.* Theriogenology, 2008. **70**(5): p. 725-745.
146. O'Rourke, K.I., et al., *Abundant PrPCWD in tonsil from mule deer with preclinical chronic wasting disease.* Journal of Veterinary Diagnostic Investigation, 2003. **15**(4): p. 320-323.
147. Argue, C.K., et al., *Epidemiology of an outbreak of chronic wasting disease on elk farms in Saskatchewan.* Canadian Veterinary Journal-Revue Veterinaire Canadienne, 2007. **48**(12): p. 1241-1248.
148. Keane, D.P., et al., *Chronic wasting disease in a Wisconsin white-tailed deer farm.* Journal of Veterinary Diagnostic Investigation, 2008. **20**(5): p. 698-703.
149. Miller, M.W., N.T. Hobbs, and S.J. Tavener, *Dynamics of prion disease transmission in mule deer.* Ecological Applications, 2006. **16**(6): p. 2208-2214.

150. Miller, M.W. and M.A. Wild, *Epidemiology of chronic wasting disease in captive white-tailed and mule deer.* Journal of Wildlife Diseases, 2004. **40**(2): p. 320-327.
151. Miller, M.W., M.A. Wild, and E.S. Williams, *Epidemiology of chronic wasting disease in captive Rocky Mountain elk.* Journal of Wildlife Diseases, 1998. **34**(3): p. 532-538.
152. Miller, M.W. and E.S. Williams, *Horizontal prion transmission in mule deer.* Nature, 2003. **425**(6953): p. 35-36.
153. Dube, C., et al., *Retrospective investigation of chronic wasting disease of cervids at the Toronto Zoo, 1973-2003.* Canadian Veterinary Journal-Revue Veterinaire Canadienne, 2006. **47**(12): p. 1185-1193.
154. Osnas, E.E., et al., *Spatial and temporal patterns of chronic wasting disease: fine-scale mapping of a wildlife epidemic in Wisconsin.* Ecological Applications, 2009. **19**(5): p. 1311-1322.
155. Grear, D.A., et al., *Demographic patterns and harvest vulnerability of chronic wasting disease infected white-tailed deer in Wisconsin.* Journal of Wildlife Management, 2006. **70**(2): p. 546-553.
156. Joly, D.O., et al., *Spatial epidemiology of chronic wasting disease in Wisconsin white-tailed deer.* Journal of Wildlife Diseases, 2006. **42**(3): p. 578-588.
157. Miller, M.W. and M.M. Conner, *Epidemiology of chronic wasting disease in free-ranging mule deer: Spatial, temporal, and demographic influences on observed prevalence patterns.* Journal of Wildlife Diseases, 2005. **41**(2): p. 275-290.
158. Miller, M.W., et al., *Epizootiology of chronic wasting disease in free-ranging cervids in Colorado and Wyoming.* Journal of Wildlife Diseases, 2000. **36**(4): p. 676-690.
159. Silbernagel, E.R., et al., *Interaction among deer in a Chronic Wasting Disease endemic zone.* Journal of Wildlife Management, 2011. **75**(6): p. 1453-1461.
160. Skuldt, L.H., N.E. Mathews, and A.M. Oyer, *White-tailed deer movements in a chronic wasting disease area in south-central Wisconsin.* Journal of Wildlife Management, 2008. **72**(5): p. 1156-1160.
161. Grear, D.A., et al., *Influence of genetic relatedness and spatial proximity on chronic wasting disease infection among female white-tailed deer.* Journal of Applied Ecology, 2010. **47**(3): p. 532-540.
162. Sharp, A. and J. Pastor, *Stable limit cycles and the paradox of enrichment in a model of chronic wasting disease.* Ecological Applications, 2011. **21**(4): p. 1024-1030.
163. Almberg, E.S., et al., *Modeling routes of Chronic Wasting Disease transmission: Environmental prion persistence promotes deer population decline and extinction.* Plos One, 2011. **6**(5): p. 11.
164. Jacobson, K.H., et al., *Transport of the pathogenic prion protein through soils.* Journal of Environmental Quality, 2010. **39**(4): p. 1145-1152.
165. Heisey, D.M., et al., *Linking process to pattern: estimating spatiotemporal dynamics of a wildlife epidemic from cross-sectional data.* Ecological Monographs, 2010. **80**(2): p. 221-240.
166. Dulberger, J., et al., *Estimating Chronic Wasting Disease effects on mule deer recruitment and population growth.* Journal of Wildlife Diseases, 2010. **46**(4): p. 1086-1095.
167. Krumm, C.E., M.M. Conner, and M.W. Miller, *Relative vulnerability of chronic wasting disease infected mule deer to vehicle collisions.* Journal of Wildlife Diseases, 2005. **41**(3): p. 503-511.
168. Farnsworth, M.L., et al., *Human land use influences chronic wasting disease prevalence in mule deer.* Ecological Applications, 2005. **15**(1): p. 119-126.
169. Schauber, E.M. and A. Woolf, *Chronic wasting disease in deer and elk: a critique of current models and their application.* Wildlife Society Bulletin, 2003. **31**(3): p. 610-616.
170. Wasserberg, G., et al., *Host culling as an adaptive management tool for chronic wasting disease in white-tailed deer: a modelling study.* Journal of Applied Ecology, 2009. **46**(2): p. 457-466.
171. Habib, T.J., et al., *Modelling landscape effects on density-contact rate relationships of deer in eastern Alberta: Implications for chronic wasting disease.* Ecological Modelling, 2011. **222**(15): p. 2722-2732.
172. Kjaer, L.J., E.M. Schauber, and C.K. Nielsen, *Spatial and temporal analysis of contact rates in female white-tailed deer.* Journal of Wildlife Management, 2008. **72**(8): p. 1819-1825.
173. Cullingham, C.I., et al., *Broad and fine-scale genetic analysis of white-tailed deer populations: estimating the relative risk of chronic wasting disease spread.* Evolutionary Applications, 2011. **4**(1): p. 116-131.
174. Cullingham, C.I., et al., *Multiscale population genetic analysis of mule deer (Odocoileus hemionus hemionus) in western Canada sheds new light on the spread of chronic wasting disease.* Canadian Journal of Zoology-Revue Canadienne De Zoologie, 2011. **89**(2): p. 134-147.
175. Kelly, A.C., et al., *Utilizing disease surveillance to examine gene flow and dispersal in white-tailed deer.* Journal of Applied Ecology, 2010. **47**(6): p. 1189-1198.
176. Schauber, E.M., D.J. Storm, and C.K. Nielsen, *Effects of joint space use and group membership on contact rates among white-tailed deer.* Journal of Wildlife Management, 2007. **71**(1): p. 155-163.
177. Conner, M.M. and M.W. Miller, *Movement patterns and spatial epidemiology of a prion disease in mule deer population units.* Ecological Applications, 2004. **14**(6): p. 1870-1881.
178. Webb, S.L., S. Demarais, and D.G. Hewitt, *Size of home ranges and movements determine size and configuration of management units and potential spread of disease white-tailed deer (Odocoileus virginianus).* Southwestern Naturalist, 2010. **55**(4): p. 488-492.

179. Frost, C.J., et al., *Probabilistic movement model with emigration simulates movements of deer in Nebraska, 1990-2006.* Ecological Modelling, 2009. **220**(19): p. 2481-2490.
180. Farnsworth, M.L., et al., *Linking chronic wasting disease to mule deer movement scales: A hierarchical Bayesian approach.* Ecological Applications, 2006. **16**(3): p. 1026-1036.
181. VerCauteren, K.C., et al., *Elk use of wallows and potential chronic wasting disease transmission.* Journal of Wildlife Diseases, 2007. **43**(4): p. 784-788.
182. Vercauteren, K.C., et al., *Fence-line contact between wild and farmed cervids in Colorado: Potential for disease transmission.* Journal of Wildlife Management, 2007. **71**(5): p. 1594-1602.
183. Vercauteren, K.C., et al., *Fence-line contact between wild and farmed white-tailed deer in Michigan: Potential for disease transmission.* Journal of Wildlife Management, 2007. **71**(5): p. 1603-1606.
184. Georgsson, G., S. Sigurdarson, and P. Brown, *Infectious agent of sheep scrapie may persist in the environment for at least 16 years.* Journal of General Virology, 2006. **87**: p. 3737-3740.
185. Cooke, C.M., et al., *Fate of prions in soil: Detergent extraction of PrP from soils.* Environmental Science & Technology, 2007. **41**(3): p. 811-817.
186. Ma, X., et al., *Adsorption of pathogenic prion protein to quartz sand.* Environmental Science & Technology, 2007. **41**(7): p. 2324-2330.
187. Maddison, B.C., et al., *The interaction of ruminant PrP(Sc) with soils is influenced by prion source and soil type.* Environmental Science & Technology. **44**(22): p. 8503-8508.
188. Saunders, S.E., et al., *Replication efficiency of soil-bound prions varies with soil type.* Journal of Virology, 2011. **85**(11): p. 5476-5482.
189. Johnson, C.J., et al., *Prions adhere to soil minerals and remain infectious.* Plos Pathogens, 2006. **2**(4): p. 296-302.
190. Johnson, C.J., et al., *Oral transmissibility of prion disease is enhanced by binding to soil particles.* Plos Pathogens, 2007. **3**(7): p. 874-881.
191. Hinckley, G.T., et al., *Persistence of pathogenic prion protein during simulated wastewater treatment processes.* Environmental Science & Technology, 2008. **42**(14): p. 5254-5259.
192. Nichols, T.A., et al., *Detection of protease-resistant cervid prion protein in water from a CWD-endemic area.* Prion, 2009. **3**(3): p. 171-183.
193. Russo, F., et al., *Pathogenic prion protein is degraded by a manganese oxide mineral found in soils.* Journal of General Virology, 2009. **90**(1): p. 275-280.
194. Walter, W.D., et al., *Soil clay content underlies prion infection odds.* Nature Communications, 2011. **2**: p. 6.
195. Cranmer, M. and T. McChesney, *Chronic wasting disease: Risks to hunters and consumers of deer and elk meat.* Neurotoxicology, 2003. **24**(2): p. 313-314.
196. Belay, E.D., et al., *Monitoring the occurrence of emerging forms of Creutzfeldt-Jakob disease in the United States.* Neurology, 2003. **60**(2): p. 176-181.
197. Belay, E.D., et al., *Chronic wasting disease and potential transmission to humans.* Emerging Infectious Diseases, 2004. **10**(6): p. 977-984.
198. Belay, E.D. and L.B. Schonberger, *The public health impact of prion diseases.* Annual Review of Public Health, 2005. **26**: p. 191-212.
199. Garruto, R.M., et al., *Risk behaviors in a rural community with a known point-source exposure to chronic wasting disease.* Environmental Health, 2008. **7**: 31, 6 pp.
200. Anderson, C.A., et al., *Colorado surveillance program for chronic wasting disease transmission to humans - Lessons from 2 highly suspicious but negative cases.* Archives of Neurology, 2007. **64**(3): p. 439-441.
201. Belay, E.D., et al., *Creutzfeldt-Jakob disease in unusually young patients who consumed venison.* Archives of Neurology, 2001. **58**(10): p. 1673-1678.
202. MaWhinney, S., et al., *Human prion disease and relative risk associated with chronic wasting disease.* Emerging Infectious Diseases, 2006. **12**(10): p. 1527-1535.
203. Kong, Q., et al., *Transmissibility of chronic wasting disease of Elk and deer to humans.* Journal of Neuropathology and Experimental Neurology, 2004. **63**(5): p. 515-515.
204. Race, B., et al., *Susceptibilities of nonhuman primates to Chronic Wasting Disease.* Emerging Infectious Diseases, 2009. **15**(9): p. 1366-1376.
205. Abrams, J.Y., et al., *Travel history, hunting, and venison consumption related to prion disease exposure, 2006-2007 Food Net population survey.* Journal of the American Dietetic Association, 2011. **111**(6): p. 858-863.
206. Coss, A.M., et al., *Exploring the zoonotic potential of chronic wasting disease in Wyoming: Creating a hunter registry*. In: Proceedings of The 131st Annual Meeting of the American Public Health Association, San Francisco, CA, November 15-19, 2003: Abstract #70648.
207. Angers, R.C., et al., *Prion strain mutation determined by prion protein conformational compatibility and primary structure.* Science, 2010. **328**(5982): p. 1154-1158.
208. Heberlein, T.A., *"Fire in the Sistine chapel": how Wisconsin responded to Chronic Wasting Disease.* Human Dimensions of Wildlife, 2004. **9**: p. 165-179.

209. Needham, M.D. and J.J. Vaske, *Hunter perceptions of similarity and trust in wildlife agencies and personal risk associated with chronic wasting disease.* Society & Natural Resources, 2008. **21**(3): p. 197-214.
210. Needham, M.D., et al., *Hunting specialization and its relationship to participation in response to chronic wasting disease.* Journal of Leisure Research, 2007. **39**(3): p. 413-437.
211. Vaske, J.J., et al., *Chronic wasting disease in Wisconsin: hunter behavior, perceived risk and agency trust.* Human Dimensions of Wildlife, 2004. **9**(3): p. 193-209.
212. Vaske, J.J. and K.M. Lyon, *CWD Prevalence, Perceived Human Health Risks, and State Influences on Deer Hunting Participation.* Risk Analysis, 2011. **31**(3): p. 488-496.
213. Stafford, N.T., et al., *Hunter and nonhunter beliefs about chronic wasting disease in Wisconsin.* Journal of Wildlife Management, 2007. **71**(5): p. 1739-1744.
214. Holsman, R.H. and J. Petchenik, *Predicting deer hunter harvest behavior in Wisconsin's chronic wasting disease eradication zone.* Human Dimensions of Wildlife, 2006. **11**(3): p. 177-189.
215. Koval, M.H., *Support for lethal wildlife management in Michigan: Results from the 1999 and 2000 Resource Attitudes in Michigan surveys.* Master of Science thesis, Department of Fisheries and Wildlife, Michigan State University: East Lansing, Michigan, 2002. 133 pp.
216. Vaske, J.J., et al., *Potential for conflict index: Hunters' responses to chronic wasting disease.* Wildlife Society Bulletin, 2006. **34**(1): p. 44-50.
217. Wallmo, K., *Economic choice modeling: the use of social preference data to inform white-tailed deer management in Michigan*. Doctor of Philosophy dissertation, Department of Fisheries and Wildlife, Michigan State University: East Lansing, Michigan, 2003. 184 pp.
218. Vaske, J.J., et al., *Information sources and knowledge about chronic wasting disease in Colorado and Wisconsin.* Human Dimensions of Wildlife, 2006. **11**(3): p. 191-202.
219. Eschenfelder, K.R. and C.A. Miller, *Examining the role of Web site information in facilitating different citizen-government relationships: A case study of state Chronic Wasting Disease Web sites.* Government Information Quarterly, 2007. **24**(1): p. 64-88.
220. Diefenbach, D.B., C.S. Rosenberry, and R.C. Boyd, *From the field: Efficacy of detecting chronic wasting disease via sampling hunter-killed white-tailed deer.* Wildlife Society Bulletin, 2004. **32**(1): p. 267-272.
221. Conner, M.M., C.W. McCarty, and M.W. Miller, *Detection of bias in harvest-based estimates of chronic wasting disease prevalence in mule deer.* Journal of Wildlife Diseases, 2000. **36**(4): p. 691-699.
222. Nusser, S.M., et al., *Sampling considerations for disease surveillance in wildlife populations.* Journal of Wildlife Management, 2008. **72**(1): p. 52-60.
223. Walsh, D.P. and M.W. Miller, *A weighted surveillance approach for detecting Chronic Wasting Disease foci.* Journal of Wildlife Diseases, 2010. **46**(1): p. 118-135.
224. Walter, W.D., et al., *Factors affecting space use overlap by white-tailed deer in an urban landscape.* International Journal of Geographical Information Science, 2011. **25**(3): p. 379-392.
225. Blanchong, J.A., et al., *Landscape genetics and the spatial distribution of chronic wasting disease.* Biology Letters, 2008. **4**(1): p. 130-133.
226. Long, E.S., et al., *Influence of roads, rivers, and mountains on natal dispersal of white-tailed deer.* Journal of Wildlife Management, 2010. **74**(6): p. 1242-1249.
227. Diefenbach, D.R., et al., *Modeling distribution of dispersal distances in male white-tailed deer.* Journal of Wildlife Management, 2008. **72**(6): p. 1296-1303.
228. Long, E.S., et al., *Forest cover influences dispersal distance of white-tailed deer.* Journal of Mammalogy, 2005. **86**(3): p. 623-629.
229. Clements, G.M., et al., *Movements of white-tailed deer in riparian habitat: Implications for infectious diseases.* Journal of Wildlife Management, 2011. **75**(6): p. 1436-1442.
230. Oyer, A.M., N.E. Mathews, and L.H. Skuldt, *Long-distance movement of a white-tailed deer away from a chronic wasting disease area.* Journal of Wildlife Management, 2007. **71**(5): p. 1635-1638.
231. Conner, M.M., et al., *A meta-BACI approach for evaluating management intervention on chronic wasting disease in mule deer.* Ecological Applications, 2007. **17**(1): p. 140-153.
232. Krumm, C.E., et al., *Mountain lions prey selectively on prion-infected mule deer.* Biology Letters, 2010. **6**(2): p. 209-211.
233. Sargeant, G.A., D.C. Weber, and D.E. Roddy, *Implications of Chronic Wasting Disease, cougar predation, and reduced recruitment for elk management.* Journal of Wildlife Management, 2011. **75**(1): p. 171-177.
234. Wild, M.A., et al., *The role of predation in disease control: A comparison of selective and nonselective removal on prion disease dynamics in deer.* Journal of Wildlife Diseases, 2011. **47**(1): p. 78-93.
235. Van Deelen, T.R., et al., *Effects of earn-a-buck and special antlerless-only seasons on Wisconsin's deer harvests.* Journal of Wildlife Management, 2010. **74**(8): p. 1693-1700.
236. Blanchong, J.A., et al., *White-tailed deer harvest from the chronic wasting disease eradication zone in south-central Wisconsin.* Wildlife Society Bulletin, 2006. **34**(3): p. 725-731.
237. Thompson, A.K., M.D. Samuel, and T.R. Van Deelen, *Alternative feeding strategies and potential disease transmission in Wisconsin white-tailed deer.* Journal of Wildlife Management, 2008. **72**(2): p. 416-421.

238. Brown, R.D. and S.M. Cooper, *The nutritional, ecological, and ethical arguments against baiting and feeding white-tailed deer.* Wildlife Society Bulletin, 2006. **34**(2): p. 519-524.
239. Johnson, C.J., et al., *Degradation of the disease-associated prion protein by a serine protease from lichens.* Plos One, 2011. **6**(5): p. 12.
240. Rosatte, R., et al., *Elk restoration in Ontario, Canada - Infectious disease management strategy, 1998-2001.* Annals of the New York Academy of Sciences, 2002. **969**: p. 358-365.

Appendix B.

Regulations for Importation Of Privately Owned Cervids into Michigan

1. Cervids to be imported:

- Must originate from a state that has NOT had a Chronic Wasting Disease (CWD) positive cervid in a privately owned herd within the past 5 years or a CWD positive cervid in any free ranging deer at any time and shall NOT originate from a herd that is within a 50 mile radius from any positive CWD case at any time. Imports from states which have had a CWD case in a privately owned herd more than 5 years prior to importation will be reviewed on a case by case basis.

- Must have continuously resided in a state that has not had a CWD positive cervid in a privately owned herd within the past 5 years or a CWD positive cervid in any free ranging deer at any time.

- Herd has attained CWD "Certified" status and the current herd owner has demonstrated compliance with the program for at least 36 months. The state of origin's CWD certification program standards must be comparable to or exceed the Michigan Department of Agriculture and Rural Development's (MDARD) standards and MDARD will confirm this prior to approval of importation.

- Must originate from a herd that is bovine Tuberculosis (TB) accredited.

- Must, if one year of age or older, have a negative brucellosis test within 30 days prior to importation or originate from a certified Brucellosis free herd.

- Must have official identification as required by MDARD and the Michigan Department of Natural Resources (MDNR) prior to entry, including all original official identification, and bear electronic ID with a reader available.

- Must have an Official Interstate Health Certificate or Certificate of Veterinary Inspection signed by an accredited veterinarian within 30 days of importation.

- The owner/manager of the herd of origin must not have been convicted of violations relating to captive cervid production compliance in this or any other herd.

2. Facility applying for importation:

- Herd has attained CWD "Certified" status and the current herd owner has demonstrated compliance with the program for at least 36 months.

- Herd must be bovine Tuberculosis (TB) accredited.

- Must be compliant with all MDNR's registration requirements stated in the *Privately Owned Cervidae Producers Marketing Act; Act 190 of 2000, as amended*, and the *Operation Standards For Registered Privately Owned Cervidae Facilities.*

- Facility must use electronic ID for tracking animals and have readers available.

- The owner/manager of the importing herd must not have been convicted of violations relating to captive cervid production compliance in this or any other herd.

3. Maximum quantities to be imported:

At the time of application for importation, each animal will be counted toward maximum numbers, this includes any fawns already born from does requested to be imported. However, if the doe is pregnant or fawns during the application period, she and those offspring will be counted as one animal for the length of the application period. If the importation request is approved, the doe may be imported pregnant or with her fawns and will only be counted as one animal against the maximum number allowed. The fawns from this doe will be tracked and held to the same post-importation movement requirements as the doe. If the importation is denied on a pregnant doe, future requests for importation must be submitted for each animal, including her fawns and they will count toward the maximum number allowed.

The maximum number for importation at an individual facility is five animals per year and 10 animals in a five year period. This is regardless of the sex of the animals imported.

4. Movement of cervids after importation:

- All cervids must stay in the importing herd for a minimum of two years, and the facility must maintain their CWD Certified status during that time.

- After two years, cervids may only be moved to another facility holding a full registration and CWD Certified status.

- Five years after importation, cervids may be moved to a herd with a ranch registration.

- **Owner must acquire a permit in writing from MDARD prior to any movement of the cervid to a new facility, whether in Michigan or to an out of state location.**

5. CWD testing requirements:

- Upon the death or harvest of the imported cervid, the owner must notify MDARD immediately and submit the appropriate testable sample for CWD testing.

- Cervids moved to ranch facilities must retain official identification and testable samples must be submitted for CWD testing upon death of the animal unless environmental conditions prevent locating the animal at the time of death.

- MDARD will consider modification of these requirements as CWD testing methods for live animals are developed and approved by USDA.

6. Fees:

- Costs associated with this approval process, including site visit travel expenses will be charged to the applicant, regardless of whether the importation request is approved or denied.

7. Review:

- This regulation must be reviewed on an annual basis and may be amended.

All of the above requirements must be met and agreed upon in order to import cervids into the state of Michigan. MDARD staff will complete verification of the requirements.

Appendix C.

Chronic Wasting Disease Decontamination Guidelines For Privately Owned Cervid Facilities

CWD is an infectious prion disease. It is transmitted between animals by direct contact with infectious saliva, respiratory aerosols, urine, and feces. CWD is also indirectly transmitted from contaminated items in the environment such as soil, where it may persist for many years. Once a CWD positive animal is identified on a premises, the Chronic Wasting Disease Response plan indicates that all cervids will be depopulated from the positive facility. Cleaning and Disinfection of the premises will occur after the depopulation has been completed to minimize environmental contamination with the CWD organism. A written premises plan will be developed by the State Veterinarian, with possible assistance from federal veterinarians, and provided to the owner of the premises for agreement. The guidelines below provide a basic framework for the development of these plans.

There are no guidelines that guarantee the total and complete elimination or inactivation of the infectious agent. The methods listed below are the most effective procedures to reduce prion levels and activity based on current information. They have been adapted from the Animal and Plant Health Inspection Service (APHIS) guidelines for decontamination of CWD affected premises. These procedures may be altered if new information becomes available.

Principals/Approach:

- The primary method(s) of transmission and the time from infection to shedding are not known. Therefore, animals may shed the CWD agent into the environment prior to the onset of clinical disease.
- The agent may be shed into the environment with saliva, urine, feces, or with fluids/placenta at the time of parturition.
- Prions are resistant to breakdown in the environment (i.e. are resistant to exposure to sunlight, freezing, desiccation, etc.) but may slowly break down with time.
- Decontamination procedures will be directed at portions of the facility or items most likely to harbor the agent. Areas of greatest contamination are related to areas where animals (particularly positive animals) have resided. These areas should be identified by using the following:

  - Assessment of the facility in detail to document areas of animal congregation or particular movement patterns.

  - Characterization of the entire facility in terms of concentration of animals over time. This includes identification of fence lines (past and present), pens, corrals or handling facilities, watering and feeding areas (including natural water sources), points of concentration in a landscape (i.e. sheltered areas, wood lots etc.), drainage areas, and calving areas.

- Identification of the distribution of the known positive or suspicious animals relative to the areas of animal concentration. In the case of clinical animals, identify those areas where they resided during the time they were clinical.

- Consideration of the physical nature of surfaces as well as topography and drainage of the area that might create concentration of the agent.

Categorization of Premises:
Premises will be categorized as premises with "No to Minimal Environmental Contamination" or "Moderate to Severe Environmental Contamination". These assessments will be performed by MDARD veterinarians, with possible assistance from USDA APHIS veterinarians.

Factors used in decision-making:
1) Origins of the positive animal(s); born to the premises or introduced
2) Herd history verified through records to adequately provide confidence in the herd status with regard to CWD (i.e. degree of certainty, or uncertainty, in relation to possible unreported cases.
3) The number of CWD cases (clinical and preclinical) originating from or occurring over time on a premises.

Basic Definitions for Categories

- "No to Minimal Environmental Contamination" – A premises where there is little evidence that there has been transmission on the premises and there is no evidence of longstanding infection of the herd. The number of cases is minimal (3 or less) and history/records indicate that the animals likely contracted the disease on another premises (i.e. trace animals). The animals are preclinical at the time of CWD diagnosis or are early in the clinical course of the disease.
- "Moderate to Severe Environmental Contamination" - Those premises where there is evidence that transmission of CWD has occurred and environmental contamination of the premises is likely; OR, Those premises where a positive animal that was likely exposed in another herd and dies of CWD or is euthanized late in the clinical course of the disease (i.e. animals are not removed from the herd while they are preclinical or early in the clinical course of the disease).

I. Response for Premises categorized as "No to Minimal Environmental Contamination"

Pastures

- Intensive measures are not required

Dry lot – Where CWD-positive animals have been held in close confinement

- Remove all bedding, manure, feed, or other organic material. Bury deeply or compost (to reduce the volume) the removed material in areas not accessed by domestic animals or wildlife. Composted material should be buried deeply, incinerated, or chemically digested after composting is complete.

Nonearth Surfaces
(These include cement, wood, metal, tools, equipment, instruments, etc.)

- Remove all organic material or items (wooden feed bunks etc.) and incinerate (by high temperature incineration methods if possible) or chemically digest the items or materials.

- Clean and wash surfaces and other items using hot water and detergent.

- Allow all surfaces, tools, and equipment to dry completely before disinfecting and sanitizing using the following methods:

    1. Autoclave instruments, small tools, and other items at 136°C (277°F) for 1 hour when possible. Pretreating by soaking in 4% sodium hydroxide will enhance the effectiveness of autoclaving.

    2. To clean dry surfaces, apply sodium hypochlorite solution with 2% available chlorine (equivalent to about 20,000 ppm available chlorine: 50 oz. [6-1/4 cups] household bleach in 1 gal water) at room temperature (at least 18.3°C [65°F]) for 1 hour. Immerse and soak items if possible. Spray large items that cannot be immersed. Sodium hypochlorite (NaOCl) is household chlorine bleach (Clorox®) and is commonly available as a 5.25% solution. Concentrated solutions of NaOCl are very corrosive to metals. For materials that are susceptible to corrosion, after an hour of treatment, rinse surface areas with fresh water taking care not to recontaminate the item. For materials not subject to corrosion, the solution may be left on for a longer period of time. Or

    3. For environmental purposes, use this disinfection method when the preceding methods are not available: Expose dry surfaces by applying 1-molar solution of sodium hydroxide (approximately 4-percent solution [5 oz. sodium hydroxide dissolved in l gal water]) at room temperature (at least 18.3°C [65°F) for at least 1 h. Synonyms for sodium hydroxide are caustic soda, soda lye, and sodium hydrate. NaOH is a white brittle deliquescent solid that dissolves readily in water to form a strong alkaline and caustic solution and is used as an alkalinizing agent. Sodium hydroxide is very caustic and in solution is EXTREMELY CORROSIVE. For environmental reasons, only use this disinfection method when the preceding method is not available.

Precautions: Professional judgment should be exercised in the choice and use of disinfectants. All disinfectants are hazardous to humans, animals, and the environment. Label directions should be carefully read and followed. If corrosive disinfectants are used directly on metal items, the items must be thoroughly rinsed with fresh water to minimize damage.

Disinfectants, especially in concentrated form, may irritate the skin, eyes, and respiratory system. Protective equipment such as coveralls, rubber boots, rubber gloves, masks or respirators, and eye protection should be worn during the mixing and application of some disinfectants. If areas of the body are exposed directly to a disinfectant, they should be washed thoroughly with water. MDARD employees should notify the State Veterinarian if excessive human or animal exposure to disinfectants occurs or if there is an accidental release into the environment.

Fencing Requirements
Fences should be maintained to prevent the ingress of free-ranging cervids for at least 5 years, or a longer time frame mutually agreed upon by the State Veterinarian and the producer. Premises may be restocked with non-cervid species immediately following decontamination.

II. Response for Premises categorized as "Moderate to Severe Environmental Contamination"

Pastures

- Effective inactivation of the agent will destroy the forage and should only be considered where exclusion of animals from high use areas is not an option. These will be approached on a case-by-case basis.
- Small pastures where CWD positive animals have resided or particular areas in a pasture where animals are known to have congregated may be treated as dry lots in some cases.

Dry lot – Where CWD-positive or CWD-exposed animals have been held in close confinement (this includes but is not limited to corrals, pens, stalls, and alleyways or pathways)

- Remove all bedding, manure, feed, or other organic material. This material may be buried deeply, incinerated, chemically digested, or composted (to reduce the volume). If material is composted, it should be done in an area inaccessible to domestic animals or wildlife. Composted material should be buried deeply, incinerated, or chemically digested after composting is complete.

- Remove the top 1–2 inches of soil. The soil removed may be buried deeply or incinerated at high temperatures.

Nonearth Surfaces
(These include cement, wood, metal, tools, equipment, instruments, etc.)

- Remove all organic material or items (wooden feed bunks etc.) and bury deeply, or incinerate (by high temperature incineration methods if possible) or chemically digest the items or materials.

- Clean and wash surfaces non-organic and other impermeable items using hot water and detergent.

- Allow all surfaces, tools, and equipment to dry completely before disinfecting and sanitizing using the following methods:

1. Autoclave instruments, small tools, and other items at 136°C (277°F) for 1 hour when possible. Pretreating by soaking in 4% sodium hydroxide will enhance the effectiveness of autoclaving.

2. To clean dry surfaces, apply sodium hypochlorite solution with 2% available chlorine (equivalent to about 20,000 ppm available chlorine: 50 oz. [6-1/4 cups] household bleach in 1 gal water) at room temperature (at least 18.3°C [65°F]) for 1 hour. Immerse and soak items if possible. Spray large items that cannot be immersed. Sodium hypochlorite (NaOCl) is household chlorine bleach (Clorox®) and is commonly available as a 5.25% solution. Concentrated solutions of NaOCl are very corrosive to metals. For materials that are susceptible to corrosion, after an hour of treatment, rinse surface areas with fresh water taking care not to recontaminate the item. For materials not subject to corrosion, the solution may be left on for a longer period of time. Or,

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